Cardiac Emergencies Workshop

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Faculty/Presenter Disclosure

- **Faculty:** Dr. Yolande Westra & Dr. Po Kee Cheung

- **Relationships with commercial interests:**
  - Speaker Honoraria with AstraZeneca & Bayer (Dr. Cheung)
  - Participating in Clinical Trial with Pegasus Trial & Regulate PCI Trial (Dr. Cheung)
Objectives

1. Discuss the common causes of cardiac arrest
2. How to Investigate and Manage catastrophic chest pain
3. How to investigate cardiac arrest due to life threatening arrhythmia.
4. Review the pathophysiology of cardiac tamponade
Case 1

• 49 year old man presented with acute onset of severe chest pain around 2 am
• Woke up from sleep
• Seen in Grande Prairie Hospital (460 km from Edmonton) at 3 am

Past Health: Not significant
Family History: Not significant
EKG on presentation
In ER

- Given ASA, NTG, O2 and Morphine
Developed This
What would you do?

What drugs would you give?
Would you transfer the patient ASAP?
In ER

- Given, clopidogrel, fibrinolytic TNK, followed by enoxaparin
EKG 1 hour post TNK
What Other Choices of Antiplatelets?
EKG 2 hour after TNK, recurrent CP
What Would You Do?
CARESS-IN-AMI: Primary Outcome

primary outcome (composite of all cause mortality, reinfarction, & refractory MI within 30 days) occurred significantly less often in the immediate PCI group vs. standard care/rescue PCI group.

primary end point: composite of death, reinfarction, recurrent ischemia, new or worsening CHF, or shock within 30 days

RR = 0.64, 95 CI% (0.47-0.87)
TRANSFER-AMI
Study Conclusion

• Following treatment with fibrinolytic therapy in high risk STEMI pts presenting to hospitals without PCI-capability, transfer to a PCI center to undergo coronary angiography and PCI should be initiated immediately without waiting to determine whether reperfusion has occurred.
Pathway: Triage and Transfer for PCI (in STEMI)

• Those presenting to a **non-PCI-capable facility** should be triaged to **fibrinolytic therapy or** immediate transfer for **PCI**.

• Decision depends on multiple clinical observations that allow judgment of:
  – mortality risk of the STEMI
  – risk of fibrinolytic therapy
  – duration of the symptoms when first seen
  – time required for transport to a PCI-capable facility
Pathway: Triage and Transfer for PCI (in STEMI) – (cont.)

- If primary PCI is chosen, the patient will be transferred for PCI.
- If fibrinolytic therapy is chosen, the patient will receive the agent(s) and a judgment as to whether the patient is high risk or not will be determined.
Pathway: Triage and Transfer for PCI (in STEMI) –
(cont.)

• If high risk, the patient should receive appropriate antithrombotic therapy and be moved immediately to a PCI-capable facility for diagnostic catheterization and consideration of PCI.
Triage and Transfer for PCI (in STEMI)

- Each community and each facility in that community should have an agreed-upon plan for how STEMI patients are to be treated, including:
  - which hospitals should receive STEMI patients from EMS units capable of obtaining diagnostic ECGs
  - management at the initial receiving hospital, and
  - written criteria & agreements for expeditious transfer of patients from non-PCI-capable to PCI-capable facilities
Case Study

19 year old well all along
1 year history of palpitation along with dizziness especially after heavy exertion
presyncope, no chest pain nor SOB
P/E loud systolic murmur at LSB grade 3/6 changes with valsalva
EKG NSR with LVH
ECHO
HOCM

• HCM is the most common genetic cardiovascular disorder (1 of every 500 adults) in the general population.
• The majority of patients are asymptomatic throughout life.
• The mortality of hypertrophic cardiomyopathy in the community is less than 1 percent per year.
• Hypertrophic cardiomyopathy is the most common cause of sudden death among young athletes (in US).
Sudden cardiac death

- Structural abnormalities
  - CAD
  - Cardiomyopathies
  - Myocarditis
  - ARVD
- Structurally normal heart
  - Long QT
  - Brugada
  - WPW
21 year old asymptomatic male with a father with SCD at age 45.
2nd EKG
Which of the following is most likely to be found during evaluation of this patient?

A. I[Kr] mutation
B. I[Ks] mutation
C. I[Na] mutation
D. Lamin A/C deficiency
E. Connexin 45
The Brugada Syndrome

Sudden death (due to VF) often at night or rest
Much more common in males (9:1), SE Asian men (SUDS)
J point elevation in V1-V3 and “RBBB” not always present;
exacerbated by Na+ channel block, fever, big meals,
alcohol binge, b-blockers, drugs; improved by
isoproterenol/exercise.
SCN5A mutations causing loss of $I_{Na}$ function– through
multiple mechanisms – now identified in ~20% of
affected subjects.
Other genes: GPD1L, SCN1B, calcium channel genes,
KCNE3, SCN3B
Brugada ECG

- Improved sensitivity with:
  - high precordial lead placement
  - Na+ channel blocker
Brugada Syndrome Risk Stratification

- **Higher Risk**
  - Syncope AND Spontaneous pattern
  - Percentage of Population: 10%
  - HR: 6.4

- **Intermediate Risk**
  - Spontaneous ECG pattern
  - Percentage of Population: 41%
  - HR: 2.1

- **Lower Risk**
  - Negative baseline ECG with or without syncope
  - Percentage of Population: 49%
EKG from a 25 year old male swimmer with no symptoms
17 year old rower with syncope with exertion. Prior history of refractory seizure disorder.
Holter Monitor,
Torsades de Pointe
What is the best treatment recommendation?

A) BB, ICD and athletic restriction

B) BB, ICD and no athletic restriction

C) BB and athletic restriction

D) BB and no athletic restriction
Prolonged QT syndrome

- Congenital
- Acquired
EKG from a 18 year old male runner with syncope with competition.
ARVD

A right sided cardiomyopathy characterized by
Histology: Fatty infiltration with remaining strands of myocardial fibers + thin fibrosis
Morphology: Localized or diffuse RV dilatation
± Electrical disturbances: Right precordial ECG anomalies, late potentials, ventricular arrhythmia